American Journal of Physiology:
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No. 1. JANUARY 1998

THEMES

MUCOSAL BIOLOGY

INFLAMMATION/IMMUNITY/MEDIATORS

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HORMONES AND SIGNALING

NEUROREGULATION AND MOTILITY

LIVER AND BILIARY TRACT

No. 3. MARCH 1998

AJP CENTENNIAL

THEMES

Current Concepts in Mucosal Immunity

EXPEDITED ARTICLE†

MUCOSAL BIOLOGY

INFLAMMATION/IMMUNITY/MEDIATORS

HORMONES AND SIGNALING

NEUROREGULATION AND MOTILITY

LIVER AND BILIARY TRACT

No. 4. APRIL 1998

THEMES

EXPEDITED ARTICLE†

ANNOUNCEMENTS

MUCOSAL BIOLOGY

INFLAMMATION/IMMUNITY/MEDIATORS

HORMONES AND SIGNALING

NEUROREGULATION AND MOTILITY

LIVER AND BILIARY TRACT

No. 5. MAY 1998

THEMES

MUCOSAL BIOLOGY

INFLAMMATION/IMMUNITY/MEDIATORS

HORMONES AND SIGNALING

NEUROREGULATION AND MOTILITY

LIVER AND BILIARY TRACT

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CORRIGENDA

Volume 274, May 1998
Volume 37, May 1998

Pages G923–G930: A. V. Mittur, N. Kaplowitz, E. S. Kempner, and M. Ookhtens. "Novel properties of hepatic canalicular reduced glutathione transport revealed by radiation inactivation." On p. G925, right column, the corrected sentence should read "In seven independent preparations, the inactivation of this enzyme yielded a target size of 93 ± 2 (SE) kDa, in excellent agreement with the known structure and previous radiation inactivation analyses (18)." Due to a typographical error, the value was mistakenly given as 83 ± 2 (SE) kDa in the original article.

American Journal of Physiology:
Lung Cellular and Molecular Physiology

No. 1. JANUARY 1998

RAPID COMMUNICATIONS

No. 2. FEBRUARY 1998

RAPID COMMUNICATION

No. 3. MARCH 1998

RAPID COMMUNICATIONS

No. 4. APRIL 1998

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SPECIAL COMMUNICATION

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No. 6. JUNE 1998

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Heart and Circulatory Physiology

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CARDIAC PHYSIOLOGY

VASCULAR PHYSIOLOGY

INTEGRATIVE CARDIOVASCULAR PHYSIOLOGY

No. 2. FEBRUARY 1998

CARDIAC PHYSIOLOGY

VASCULAR PHYSIOLOGY

INTEGRATIVE CARDIOVASCULAR PHYSIOLOGY

No. 3. MARCH 1998

CARDIAC PHYSIOLOGY

VASCULAR PHYSIOLOGY

INTEGRATIVE CARDIOVASCULAR PHYSIOLOGY

No. 4. APRIL 1998

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VASCULAR PHYSIOLOGY

INTEGRATIVE CARDIOVASCULAR PHYSIOLOGY

No. 5. MAY 1998

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VASCULAR PHYSIOLOGY

INTEGRATIVE CARDIOVASCULAR PHYSIOLOGY

No. 6. JUNE 1998

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VASCULAR PHYSIOLOGY

INTEGRATIVE CARDIOVASCULAR PHYSIOLOGY

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CORRIGENDA

Volume 271, October 1996
Volume 40, October 1996

Pages H1307–H1313: Hideyuki Sasaki and Phillip A. Low. "Extreme vasoreactivity of rat epineurial arterioles to vasopressin." The published paper contains erroneous data on the threshold and EC_{50} of the vasopressin dose-response studies, likely due to a large and systematic dilutional error in the preparation of the vasopressin solution. All vasopressin concentrations given in the paper in the abstract; in METHODS, RESULTS, DISCUSSION; and in the tables, figures, and legends, together with statements about "orders of magnitude" for the newly reported data in the DISCUSSION, are incorrect. The topical application studies were repeated on the nerves of 42 rats (7 concentrations; $n = 6$ for each concentration; 1 nerve per rat), and a new dose-response curve was obtained. The correct EC_{50} is 6.6×10^{-9} M being about two orders of magnitude more potent than norepinephrine [Kihara and Low. *J. Physiol. (Lond)* 422: 145–152, 1990] and approximately equipotent with endothelin [Zochodne, Ho, and Gross. *Am. J. Physiol.* 263 (*Heart Circ. Physiol.* 32): H1806–H1810, 1992; Kihara and Low. *Exp. Neurol.* 132: 180–185, 1994]. The studies on intra-arterially administered vasopressin have not been repeated and should be ignored. The conclusion of studies on interactions of vasopressin with α-adrenergic receptors are correct, since they only require that the concentrations of the agents are subthreshold. Similarly, the conclusions of the study on the effects of topically applied vasopressin and norepinephrine on nerve conduction are also correct, since they only require a high concentration of both agents. A corrected abstract appears below.

Sasaki, Hideyuki, and Phillip A. Low. Extreme vasoreactivity of rat epineurial arterioles to vasopressin. *Am. J. Physiol.* 271 (*Heart Circ. Physiol.* 40): H1307–H1313, 1996.—Vasopressin is a potent vasoconstrictor to most blood vessels but is a vasodilator to some. The role of vasopressin in the regulation of nerve blood flow (NBF) is not known. We undertook a dose-effect study of vasopressin on NBF and evaluated its interactions with α-adrenoreceptors and its effect on ischemic conduction failure. NBF was measured using microelectrode hydrogen polarography. Vasopressin was administered topically (to epineurium). Topical epineurial application of vasopressin caused a concentration-dependent reduction of NBF ($EC_{50} = 6.6 \times 10^{-9}$ M; asymptote = 73.9% NBF reduction). The topical application of subthreshold concentrations of vasopressin and norepinephrine alone resulted in no change in NBF, but combined application resulted in a dramatic reduction in NBF (72.3%). The ratio of amplitudes of muscle compound action potential evoked on proximal to distal stimulation was used as an index of the presence of an ischemic conduction block. This ratio was significantly reduced following the combined topical application of supramaximal concentrations of vasopressin and norepinephrine. These findings suggest that vasopressin is a potent neural vasoconstrictor and that vasoconstriction caused by combined vasopressin and norepinephrine can produce partial conduction block of sciatic-tibial nerve.

American Journal of Physiology:
Regulatory, Integrative and Comparative Physiology

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REGULATORY AND INTEGRATIVE PHYSIOLOGY

DEVELOPMENTAL PHYSIOLOGY

COMPARATIVE PHYSIOLOGY

No. 2. FEBRUARY 1998

INVITED REVIEW

REGULATORY AND INTEGRATIVE PHYSIOLOGY

DEVELOPMENTAL PHYSIOLOGY

COMPARATIVE PHYSIOLOGY

No. 3. MARCH 1998

INVITED REVIEW

REGULATORY AND INTEGRATIVE PHYSIOLOGY

DEVELOPMENTAL PHYSIOLOGY

COMPARATIVE PHYSIOLOGY

No. 4. APRIL 1998

INVITED REVIEW

REGULATORY AND INTEGRATIVE PHYSIOLOGY

DEVELOPMENTAL PHYSIOLOGY

COMPARATIVE PHYSIOLOGY

No. 5. MAY 1998

REGULATORY AND INTEGRATIVE PHYSIOLOGY

DEVELOPMENTAL PHYSIOLOGY

COMPARATIVE PHYSIOLOGY

No. 6. JUNE 1998

REGULATORY AND INTEGRATIVE PHYSIOLOGY

DEVELOPMENTAL PHYSIOLOGY

COMPARATIVE PHYSIOLOGY

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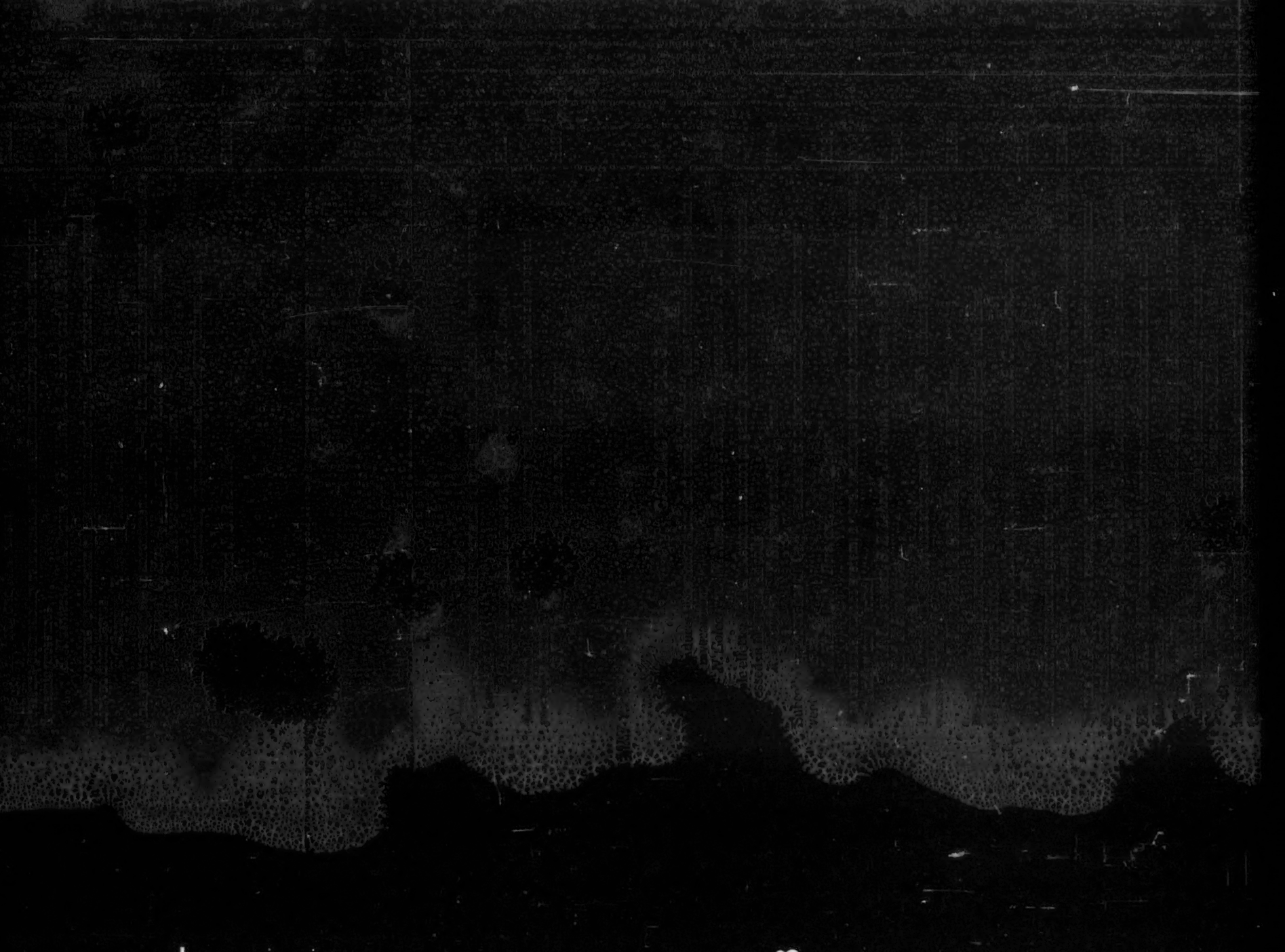
CORRIGENDA

Volume 273, August 1997
Volume 42, August 1997

Pages R568–R577: T. E. N. Jonassen, N. Marcussen, K. Haugan, H. Skyum, S. Christensen, F. Andreasen, and J. S. Petersen. "Functional and structural changes in the thick ascending limb of Henle's loop in rats with liver cirrhosis." On p. R568, the journal citation in the abstract should read as follows: *Am. J. Physiol.* 273 (*Regulatory Integrative Comp. Physiol.* 42): **R568–R577**, 1997.



American Journal of Physiology:
Renal Physiology

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INVITED REVIEW

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No. 3. MARCH 1998

RAPID COMMUNICATION

ANNOUNCEMENT

No. 4. APRIL 1998

RAPID COMMUNICATION

No. 5. MAY 1998

AJP CENTENNIAL

No. 6. JUNE 1998

INVITED REVIEW

RAPID COMMUNICATION

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Advances in Physiology Education

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A CELEBRATION OF THE CENTENNIAL OF THE *AMERICAN JOURNAL OF PHYSIOLOGY*

INNOVATIONS AND IDEAS

EDUCATIONAL EXPERIMENTS